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L1 140 (ROUGH EYE) (8A) (DROSOPHILA OR FLY OR FLIES)

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ENETIC)

=> d l2 1-4 bib ab

L2 ANSWER 1 OF 4 MEDLINE on STN
AN 2001069167 MEDLINE
DN PubMed ID: 11063696
TI A misexpression screen identifies genes that can modulate RAS1 pathway
signaling in Drosophila melanogaster.
AU Huang A M; Rubin G M
CS Department of Molecular and Cell Biology, University of California,
Berkeley, California 94720-3200, USA.
NC HG00750 (United States NHGRI NIH HHS)
SO Genetics, (2000 Nov) Vol. 156, No. 3, pp. 1219-30.
Journal code: 0374636. ISSN: 0016-6731.
Report No.: NLM-PMC1461302.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LA English
FS Priority Journals
EM 200101
ED Entered STN: 22 Mar 2001
Last Updated on STN: 20 Apr 2002
Entered Medline: 4 Jan 2001
AB Differentiation of the R7 photoreceptor cell is dependent on the Sevenless

receptor tyrosine kinase, which activates the RAS1/mitogen-activated protein kinase signaling cascade. Kinase suppressor of Ras (KSR) functions genetically downstream of RAS1 in this signal transduction cascade. Expression of dominant-negative KSR (KDN) in the developing eye blocks RAS pathway signaling, prevents R7 cell differentiation, and causes a rough eye phenotype. To identify genes that modulate RAS signaling, we screened for genes that alter RAS1/KSR signaling efficiency when misexpressed. In this screen, we recovered three known genes, Lk6, misshapen, and Akap200. We also identified seven previously undescribed genes; one encodes a novel rel domain member of the NFAT family, and six encode novel proteins. These genes may represent new components of the RAS pathway or components of other signaling pathways that can modulate signaling by RAS. We discuss the utility of gain-of-function screens in identifying new components of signaling pathways in *Drosophila*.

L2 ANSWER 2 OF 4 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
 AN 2000398081 EMBASE
 TI A misexpression screen identifies genes that can modulate RAS1 pathway signaling in *Drosophila melanogaster*.
 AU Huang, A.M.; Rubin, G.M. (correspondence)
 CS Howard Hughes Medical Institute, 545 Life Sciences Addition no. 3200, University of California, Berkeley, CA 94720-3200, United States. gerry@fruitfly.BDGP.berkeley.edu
 SO Genetics, (2000) Vol. 156, No. 3, pp. 1219-1230.
 Refs: 59
 ISSN: 0016-6731 CODEN: GENTAE
 CY United States
 DT Journal; Article
 FS 012 Ophthalmology
 021 Developmental Biology and Teratology
 022 Human Genetics
 LA English
 SL English
 ED Entered STN: 13 Dec 2000
 Last Updated on STN: 13 Dec 2000
 AB Differentiation of the R7 photoreceptor cell is dependent on the Sevenless receptor tyrosine kinase, which activates the RAS1/mitogen-activated protein kinase signaling cascade. Kinase suppressor of Ras (KSR) functions genetically downstream of RAS1 in this signal transduction cascade. Expression of dominant-negative KSR (KDN) in the developing eye blocks RAS pathway signaling, prevents R7 cell differentiation, and causes a rough eye phenotype. To identify genes that modulate RAS signaling, we screened for genes that alter RAS1/KSR signaling efficiency when misexpressed. In this screen, we recovered three known genes, Lk6, misshapen, and Akap200. We also identified seven previously undescribed genes; one encodes a novel rel domain member of the NFAT family, and six encode novel proteins. These genes may represent new components of the RAS pathway or components of other signaling pathways that can modulate signaling by RAS. We discuss the utility of gain-of-function screens in identifying new components of signaling pathways in *Drosophila*.

L2 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
 AN 2001:21028 BIOSIS
 DN PREV200100021028
 TI A misexpression screen identifies genes that can modulate RAS1 pathway signaling in *Drosophila melanogaster*.
 AU Huang, Audrey M.; Rubin, Gerald M. [Reprint author]
 CS Howard Hughes Medical Institute, University of California, 545 Life

Sciences Addition No. 3200, Berkeley, CA, 94720-3200, USA
 gerry@fruitfly.BDGP.berkeley.edu
 SO Genetics, (November, 2000) Vol. 156, No. 3, pp. 1219-1230. print.
 CODEN: GENTAE. ISSN: 0016-6731.
 DT Article
 LA English
 ED Entered STN: 3 Jan 2001
 Last Updated on STN: 12 Feb 2002
 AB Differentiation of the R7 photoreceptor cell is dependent on the Sevenless
 receptor tyrosine kinase, which activates the RAS1/mitogen-activated
 protein kinase signaling cascade. Kinase suppressor of Ras (KSR)
 functions genetically downstream of RAS1 in this signal transduction
 cascade. Expression of dominant-negative KSR (KDN) in the developing eye
 blocks RAS pathway signaling, prevents R7 cell
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 genes may represent new components of the RAS pathway or components of
 other signaling pathways that can modulate signaling by RAS. We discuss
 the utility of gain-of-function screens in identifying new components of
 signaling pathways in Drosophila.

L2 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2000:850437 CAPLUS
 DN 135:176197
 TI A misexpression screen identifies genes that can modulate RAS1 pathway
 signaling in Drosophila melanogaster
 AU Huang, Audrey M.; Rubin, Gerald M.
 CS Department of Molecular and Cell Biology, University of California,
 Berkeley, CA, 94720-3200, USA
 SO Genetics (2000), 156(3), 1219-1230
 CODEN: GENTAE; ISSN: 0016-6731
 PB Genetics Society of America
 DT Journal
 LA English
 AB Differentiation of the R7 photoreceptor cell is dependent on the Sevenless
 receptor tyrosine kinase, which activates the RAS1/mitogen-activated
 protein kinase signaling cascade. Kinase suppressor of Ras (KSR)
 functions genetically downstream of RAS1 in this signal transduction
 cascade. Expression of dominant-neg. KSR (KDN) in the developing eye
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 other signaling pathways that can modulate signaling by RAS. We discuss
 the utility of gain-of-function screens in identifying new components of
 signaling pathways in Drosophila.

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